=> d l6 1-4 all

L6 ANSWER 1 OF 4 MEDLINE on STN
AN 2000261741 MEDLINE
DN PubMed ID: 10799595
TI Yin yang 1 negatively regulates the differentiation-specific E1 promoter of human papillomavirus type 6.
AU Ai W; Narahari J; Roman A
CS Department of Microbiology and Immunology, Indiana University School of Medicine, and Walther Cancer Institute, Indianapolis, Indiana 46202-5120, USA.
NC AI31494 (NIAID)
SO Journal of virology, (2000 Jun) Vol. 74, No. 11, pp. 5198-205.
Journal code: 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200006
ED Entered STN: 6 Jul 2000
Last Updated on STN: 6 Jul 2000
Entered Medline: 27 Jun 2000
AB Human papillomavirus type 6 (HPV-6) is a low-risk HPV whose replication cycle, like that of all HPVs, is differentiation dependent. We have previously shown that CCAAT displacement protein (CDP) binds the differentiation-induced HPV-6 E1 promoter and negatively regulates its activity in undifferentiated cells (W. Ai, E. Toussaint, and A. Roman, J. Virol. 73:4220-4229, 1999). Using electrophoretic mobility shift assays (EMSAs), we now report that Yin Yang 1 (YY1), a multifunctional protein that can act as a transcriptional activator or repressor and that can also inhibit HPV replication in vitro, binds the HPV-6 E1 promoter. EMSAs, using subfragments of the promoter as competitors, showed that the YY1 binding site is located at the 5' end of the E1 promoter. When a putative YY1 site was mutated, the ability of YY1 to bind was greatly decreased. The activity of the mutated E1 promoter, monitored with the reporter gene luciferase, was threefold greater than that of the wild-type promoter, suggesting that YY1 negatively regulates HPV-6 E1 promoter activity. Nuclear extracts from differentiated keratinocytes showed decreased binding of YY1 to the wild-type promoter. Consistent with this, in differentiated keratinocytes, the activity of the transfected luciferase gene transcribed from the mutated promoter was comparable to that of the wild-type promoter; both promoters were up-regulated in differentiated keratinocytes compared to undifferentiated cells. These data suggest that YY1 functions in undifferentiated keratinocytes but not in differentiated keratinocytes. Both the wild-type and mutated promoters could be negatively regulated by overexpression of a plasmid encoding CDP. Thus, both YY1 and CDP appear to be negative regulators of the differentiation-induced HPV-6 E1 promoter and thereby the HPV life cycle. In contrast, only binding of CDP was detected using the E1 promoter of the high-risk HPV-31.
CT 3T3 Cells
Animals
Base Sequence
Binding Sites
Cell Differentiation
Cells, Cultured
DNA, Viral
*DNA-Binding Proteins: ME, metabolism
Erythroid-Specific DNA-Binding Factors
Humans
Keratinocytes: CY, cytology
Mice
Molecular Sequence Data

Mutagenesis
Nuclear Proteins: ME, metabolism
*Oncogene Proteins, Viral: GE, genetics
*Papillomavirus, Human: GE, genetics
*Promoter Regions (Genetics)
*Repressor Proteins: ME, metabolism
Research Support, U.S. Gov't, P.H.S.
*Transcription Factors: ME, metabolism
Viral Proteins: GE, genetics
YY1 Transcription Factor

CN 0 (CUTL1 protein, human); 0 (DNA, Viral); 0 (DNA-Binding Proteins); 0 (E1 protein, Human papillomavirus type 31); 0 (E1 protein, Human papillomavirus type 6); 0 (Erythroid-Specific DNA-Binding Factors); 0 (Nuclear Proteins); 0 (Oncogene Proteins, Viral); 0 (Repressor Proteins); 0 (Transcription Factors); 0 (Viral Proteins); 0 (YY1 Transcription Factor); 0 (YY1 protein, human); 0 (Yy1 protein, mouse)

L6 ANSWER 2 OF 4 MEDLINE on STN
AN 1998001334 MEDLINE
DN PubMed ID: 9343169
TI Differential effects of the splice acceptor at nucleotide 3295 of human papillomavirus type 31 on stable and transient viral replication.
AU Klumpp D J; Stubenrauch F; Laimins L A
CS Department of Microbiology-Immunology, Northwestern University Medical School, Chicago, Illinois 60611, USA.
NC F32 AI09494-01 (NIAID)
R01 CA-59655 (NCI)
SO Journal of virology, (1997 Nov) Vol. 71, No. 11, pp. 8186-94.
Journal code: 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199711
ED Entered STN: 24 Dec 1997
Last Updated on STN: 24 Dec 1997
Entered Medline: 13 Nov 1997
AB In human papillomavirus type 31 (HPV-31), the E1--E4 and E5 open reading frames are expressed from polycistronic mRNAs. The major polycistronic mRNAs which encode E1--E4 and E5 are spliced messages which utilize a splice acceptor at nucleotide (nt) 3295 (SPA3295). Our laboratory recently developed a recombinant system for the synthesis of HPVs following immortalization of primary keratinocytes with cloned HPV-31 genomes (M. G. Frattini et al., Proc. Natl. Acad. Sci. USA 93:3062-3067, 1996). These immortalized cell lines are capable of maintaining HPV-31 DNA as episomes and induce the synthesis of virions in organotypic raft culture. In this study, we used these methods to begin an analysis of the roles of E1--E4 and E5 in HPV pathogenesis by mutating the major splice at nt 3295. Mutation of SPA3295 did not significantly alter the ability of HPV-31 genomes to replicate transiently in keratinocytes, nor did the mutation affect the immortalization potential of HPV-31. However, genomes carrying the SPA3295 mutation were not stably maintained as viral episomes, and the resulting immortalized keratinocyte cell line contained multiple, integrated copies of the mutated HPV-31 DNA. Northern analysis indicated that cell lines immortalized with the mutant HPV-31 expressed transcripts which were similar in size and abundance to wild-type messages, including those transcripts which rely on utilization of SPA3295. RNase protection and reverse transcription-PCR revealed that mutation of SPA3295 resulted in the utilization of a cryptic splice acceptor at nt 3298. These data suggest that the requirements for stable maintenance of HPV genomes are more stringent than those for transient replication and that factors which define these requirement rely on the major splice acceptor at nt 3295.

CT Alternative Splicing
DNA, Viral: GE, genetics
Gene Expression Regulation, Viral
Humans
Keratinocytes: VI, virology
*Papillomavirus, Human: GE, genetics
Plasmids
RNA, Viral: GE, genetics
Research Support, Non-U.S. Gov't
Research Support, U.S. Gov't, P.H.S.
*Virus Integration
*Virus Replication
CN 0 (DNA, Viral); 0 (RNA, Viral)

L6 ANSWER 3 OF 4 MEDLINE on STN
AN 94346757 MEDLINE
DN PubMed ID: 8067697
TI Involvement of aberrant p53 expression and human papillomavirus in carcinoma of the head, neck and esophagus.
AU Lewensohn-Fuchs I; Munck-Wikland E; Berke Z; Magnusson K P; Pallesen G; Auer G; Lindholm J; Linde A; Aberg B; Rubio C; +
CS Department of Immunology, Microbiology, Pathology, Karolinska Institute, Huddinge Hospital, Sweden.
SO Anticancer research, (1994 May-Jun) Vol. 14, No. 3B, pp. 1281-5.
Journal code: 8102988. ISSN: 0250-7005.
CY Greece
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199409
ED Entered STN: 5 Oct 1994
Last Updated on STN: 6 Feb 1998
Entered Medline: 22 Sep 1994
AB Biopsies from 34 patients with cancer of the head, neck or esophagus, 2 laryngeal papillomas, and 2 normal tonsils were analysed for human papillomavirus (HPV), Epstein Barr virus (EBV) genomes and mutated or elevated levels of p53. In 4 biopsies p53 was also analysed by DNA sequencing. HPV type 31 was found in one laryngeal cancer with normal p53 and HPV type 16 in two tonsil cancers with aberrant p53 expression. EBV was detected by PCR in 11 biopsies, but in situ hybridisation and immunohistochemistry, did not confirm this finding. Aberrant p53 expression was observed in approximately half of the tumours. These results support the involvement of both aberrant p53 expression and HPV in the aetiology of squamous cell carcinoma of the head and neck.
CT Base Sequence
*Carcinoma, Squamous Cell: ET, etiology
Carcinoma, Squamous Cell: GE, genetics
Carcinoma, Squamous Cell: VI, virology
*Esophageal Neoplasms: ET, etiology
Esophageal Neoplasms: GE, genetics
Esophageal Neoplasms: VI, virology
Follow-Up Studies
*Head and Neck Neoplasms: ET, etiology
Head and Neck Neoplasms: GE, genetics
Head and Neck Neoplasms: MI, microbiology
Head and Neck Neoplasms: VI, virology
Herpesvirus 4, Human: IP, isolation & purification
Humans
Molecular Sequence Data
Mutation
*Papillomavirus, Human: IP, isolation & purification
Polymerase Chain Reaction
Research Support, Non-U.S. Gov't

*Tumor Suppressor Protein p53: AN, analysis
CN 0 (Tumor Suppressor Protein p53)

L6 ANSWER 4 OF 4 MEDLINE on STN
AN 94172830 MEDLINE
DN PubMed ID: 8126914
TI Detection of human papillomavirus DNA and state of p53 gene in Japanese penile cancer.
AU Suzuki H; Sato N; Kodama T; Okano T; Isaka S; Shirasawa H; Simizu B; Shimazaki J
CS Department of Urology, School of Medicine, Chiba University.
SO Japanese journal of clinical oncology, (1994 Feb) Vol. 24, No. 1, pp. 1-6. Journal code: 0313225. ISSN: 0368-2811.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199404
ED Entered STN: 20 Apr 1994
Last Updated on STN: 6 Feb 1998
Entered Medline: 8 Apr 1994
AB The frequency of integration with human papillomavirus (HPV) and its genotypes in Japanese penile cancer was examined with relation to p53 gene mutations using polymerase chain reaction amplification. Tissues were obtained from 13 patients (eight from freshly frozen and five from paraffin-embedded samples). HPV DNA was detected in seven out of the 13 (54%), and their genotypes were type 16 in four, type 31 in one and type 33 in two cases. Neither HPV-detected nor -undetected tissues showed mutated alterations in exons 4-9 of p53 genes. The results suggest HPV to be, at least to some extent, involved in the oncogenesis of penile cancer, and that p53 gene mutations may not correlate with the development of penile cancer.
CT Check Tags: Male
Aged
Aged, 80 and over
Base Sequence

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=> d his

     (FILE 'HOME' ENTERED AT 09:07:39 ON 19 DEC 2006)

     FILE 'MEDLINE' ENTERED AT 09:07:53 ON 19 DEC 2006
L1         17976 S PAPILLOMAVIRUS
L2           160 S TYPE 31
L3            80 S L1 AND L2
L4           163 S CODON OPTIMIZED
L5             0 S L3 AND L4
L6             4 S L3 AND MUTATED
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